PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, November 22d, 1886.

The Vice-President, CHARLES K. MILLS, M.D., *in the Chair.*

DR. E. C. SEGUIN, of New York, read by invitation

NOTES ON THREE CASES OF TROPICAL BERI-BERI, AND ON SOME SIMILAR AMERICAN CASES.

Having been courteously invited to participate in this evening's symposium on neuritis, I bring my mite of contribution in the shape of the relation of three cases of beri-beri, which, coming from tropical parts, have been under my care. That this disease is rarely seen in our latitudes, though very common in South and Central America and in the islands of the Gulf, I infer from the fact that it has not, to my knowledge, been discussed in medical societies or in private relations in New York during the past ten or fifteen years.

My apology for relating the following cases is that they well illustrate the prominence of neural symptoms in beri-beri, and that they justify the opinion expressed by a number of observers (Dr. B. Scheube and others) that beri-beri, or kak-ke, as the affection is called in Japan, is essentially a multiple, poly, or panneuritis, occurring as an endemic infectious (?) disease in many countries of the globe in both hemispheres. That beri-beri, or kak-ke, is nothing more than a multiple neuritis I am not prepared to say, and, indeed, my experience is so extremely limited that I can only use my cases to illustrate the probable neural origin of many of the symptoms of the disease.

In looking for notes of cases of multiple neuritis occurring in my local practice I found several. One, very typical, was recorded under the title of alcoholic polyneuritis; others were included in my essays on poliomyelitis anterior,[1] as examples of that disease.

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[1] "Myelitis of the Anterior Horns; or, Spinal Paralysis of the Adult and Child." New York, 1877. (Private edition, 1874.)

These three cases were marked by the ordinary symptoms of multiple neuritis: numbness, and partial anæsthesia, irregularly distributed atrophic paralysis, with degenerative reactions ; and, besides, œdema of the extremities. I was struck by the resemblance of these cases, thus symptomatized, to mild cases of the mixed form of beri-beri. There is quite a resemblance, if not a similarity, between Cases I. and II. of the category of beri-beri and Case I. of the category of indigenous neuritis.

Another aspect in which a study of beri-beri is of interest to us is that the disease may take root and acquire considerable development in the United States. It appears that the affection has prevailed in Para, Brazil, for only a few years, perhaps only since 1878, having been brought there from Southern Brazil and Paraguay ; and, as it is not uncommon in Cuba (at least the so-called wet form), it may very well show itself in the Gulf States.

With this brief preface, I pass on to the cases themselves.

First Category: Case of Beri-beri.

Case I.—A Cuban lawyer, aged twenty-six, consulted me September 9th, 1885, with letters from Drs. Landeta and Desvernines, of Havana, of which the following is an abstract : Married man of good constitution. Never had syphilis, scrofula, or paludism : never seriously ill. In summer and autumn of 1884, dyspeptic symptoms.

Symptoms of present illness : (1) Early in November (1884), pains in lower third of both legs. (2) In a few days these parts were slightly œdematous, while the malleoli and feet remained free from swelling. (3) Simultaneously, cardiac palpitations ; rapid pulse, from 110 to 120. (4) Some muscular weakness was noticed in legs ; walk difficult and done on tip-toe, or on forward part of foot. (5) Toward end of November the pulse was steadily 120 or more beats ; large objective palpitations and souffles were noticed over cardiac area. (6) Œdema appeared in the face, invaded the whole of the legs, thighs, feet, and genitals. (7) The muscles of the thighs and legs became soft and painful to touch; there seemed to be intramuscular œdema. Toward the end of the month patient was unable to leave the bed ; the palpitation was intense, and there was great dyspnœa. The thenar and hypothenar eminences became soft ; the patient could no longer write, or use fork, spoon, etc. Later the weakness became such that the legs could not be lifted from the bed : face was nearly twice its normal size. The patellar reflex was abolished. Some muscles showed loss of contractility under electricity (*vide infra*).

As a summary of symptoms in the fourth and fifth weeks of the disease, it may be said that there were anasarca, paralysis with electrical changes in certain muscular groups, cardiac bruits, with pulse up to 140 beats per minute, dyspnœa. At no time was there fever, and no albumin or sugar was ever found in urine.

Under the use of infusion of digitalis, cinchona, and nutritious food, these symptoms abated, and there was decided improvement by the end of December (eighth week). Under electrical treatment and sulphur baths the motor paralysis gradually passed off, and the patient was able to stand in March : steady improvement thereafter continued, the cardiac symptoms had about ceased by the end of December.

Dr. Desvernine's letter refers more particularly to the state of the heart, and to the electrical reactions. At the height of the disease the heart was enlarged, more especially the left ventricle, and the murmur heard was a mitral regurgitant one. These signs were due, Dr. Desvernines believes, to myo-cardial lesions ; the endocardium being unaffected. This view is borne out by the disappearance of the symptoms.

There was an apparent pseudo-hypertrophy of all muscles of the body (œdema). A piece of muscle removed by Duchenne's trocar showed simple atrophy of its muscular fibrillæ with intramuscular œdema and proliferation of the connective tissue. The electrical reactions were not taken during the acute period of the disease, but afterward were very fully studied, and there were found with both currents the "reactions of degeneration, characteristic of perverted nutrition in nerve and muscle."

The diagnosis made by Drs. Landeta and Desvernines was beriberi or kak-ke, of the mixed form, and Dr. Desvernines recognized in the case the grounds for the views advanced by Scheube and Balz (from studies of the disease in Japan), that it is essentially a multiple neuritis. It is interesting to add that, while the wet form (anæmic œdematous type) is well known to the physicians of Cuba and Havana, not one of several who saw this case with Dr. Landeta had seen an example of the mixed form.

When I examined Señor M. about ten months after the beginning of the attack, he presented no objective symptoms except a degree of abnormal flushing of the face. Excepting a slight quantitative reduction in contractility of the calf muscles, I noticed no abnormality in the faradic and galvanic reactions of nerve, trunk, and muscles. The pulse was slow, from 57 to 63, and the heart normal in size and sound. The patient complained of occasional vertigo, and want of power of mental concentration. He was well October 10th.

Case II.—Mr. J. G., a married man, thirty-eight years of age, a native and resident of Panama, was brought to me, for consultation, by Dr. Pio Rengifo, of New York, on November 17th, 1885. Is a tall, strongly-built man, who has enjoyed good health, except that sixteen years ago he had a single chancre with suppurating buboes, but no secondary symptoms. Had a year's treatment with Dupuytren's pills and iodide of potassium. A healthy child has been the result of marriage. In the past eight years occasional palpitations, supposed to be due to gastric disorder. Is temperate in use of stimulants. About two months ago noticed numbness in toes and fingers. This has continued as a constant symptom, affecting tips of fingers, toes, and forward part of soles ; on lying down, however, there has been a certain sense of numbness in the legs and forearms. Pains appeared later, of two kinds : first, aching pains in the calves, which were hard and tender ; second, sharp pains in spots in fingers, legs, and feet ; not, however, repeated pains in spots, with cutaneous hyperalgesia (not fulgurating pains). Knees were weak, and toes seemed cramped. Had sensation of band around toes. Had no vesical paresis, loss of sexual power, or spinal pains ; no abnormal reflexes in legs, no diplopia. In last three weeks distinct œdema of feet and legs ; has noticed that the calves measure two or three centimetres more in the evening than in the morning. During the past fortnight there has been marked improvement in power of legs, and in the paræsthesiæ.

Examination.—No objective symptoms about eyes or face. Grasp : right side, 38 and 32 degrees ; left, 31 and 22 degrees ; decidedly weak for a man of patient's muscular development and size. Sensibility is not positively diminished in fingers, but there is some confusion in estimating number of points applied ; distinguishing texture of cloths well. No analgesia. Fingers are skilful ; handwriting unchanged.

Lower extremities: no patellar reflex ; walks a little awkwardly, with slapping of the feet; but, strictly speaking, there is no ataxia. Staggers when standing with eyes closed and feet together, but with eyes open stands fairly well, even on one foot. Sensibility to æsthesiometer is dull, but he localizes impressions well; no analgesia. Those movements of toes which are controlled by interossei are limited. There is yet a trace of œdema over the tibiæ. No electrical examination.

I concurred in Dr. Rengifo's diagnosis of beri-beri or multiple neuritis, and the same opinion was expressed by Professor Charcot, who saw Mr. G. shortly after. I recently learned that entire recovery took place in three months. It may be of interest to add that Mr. G. presented complete transposition of the viscera, his heart (the sounds of which were normal) being distinctly on the right side of the sternum, and his liver on the left side; he was, however, right-handed originally.

Case III.—Señor J., aged fifty, a native and resident of Para, in Brazil, was referred to me by my friend, Dr. Charles E. Simmons, on November 11th, and is now under observation. Former health good; never had beri-beri or severe malarial fever. Subject to "bilious attacks." In August of present year had bleeding hemorrhoids for three weeks, followed by weakness of legs. This was soon followed by tingling numbness and pains in the legs, and lastly by œdema, not affecting feet. By advice of physicians, Mr. J. sailed from Para, October 27th, but instead of immediately improving, he grew worse. Soon noticed numbness and awkwardness of hands; the weakness and œdema of legs increased. In the last six days the long extensors and the long flexors of the right thumb have shown paresis, and in the last four days the same muscles on the left side have been affected. No dyspnœa, vertigo, or mental symptoms. No fever observed; urine high colored. The pains were never severe.

Examination.—Stout, well-built man; complexion pale, muddy, and tanned. No motor or sensory symptoms in eyes or face. Heart normal; no effusion in pleura. Upper extremities: all voluntary movements present except extension and flexion of phalanges of both thumbs; the two long extensors on both sides are completely paralyzed, and the long flexors partially so. The thumbs hang as do fingers in certain cases of lead paralysis. The left extensor indicis is weak. The abductor pollicis longus, and the small hand muscles are normal on both sides. Opposition well done. Supinators strong. No ataxia. Grasp: right and left, each 25 degrees on dynamometer. Sensibility is normal to æsthesiometer on finger-tips; but, by pricking, a limited area of analgesia (incomplete) is found over the left metacarpus, in the ulnar territory. Temperature well perceived. Lower extremities: the walk is somewhat feeble, but not pathological in type; can stand upon his toes, and equilibrium with eyes open or closed is nearly perfect. No ataxia. Patellar reflex abnormal on both sides. Both legs present a soft œdema in their inferior half; the calves are large but flabby. No swelling distal of ankle-joints. All voluntary movements of legs, feet, and toes can be performed; though extension and flexion of toes are done feebly. The great toes are not as much affected as are the thumbs. Sensibility is slightly dull to touch (a numb feeling) as high as the umbilicus; but the only zone of actual analgesia is an outer (fibular) side of left leg in its lower third. Temperature well perceived. Urine is dense, deposits urates; without albumin; a few small hyaline casts. Summary of electrical examination: Arms, no faradic or galvanic nerve reaction in extensor primi et secundi internodii pollicis on either side. These muscles do not respond to faradism, while galvanism yields a slow contraction, with marked tendency to tetanus: CaCC = AnCC or CaCC < AnCC. In flexor longus pollicis (both sides) there is no nerve reaction, and only a faint oscillatory C, with a strong faradic current applied directly to muscle. Galvanism gives a slow contraction: CaCC = AnCC, and the latter tending to tetanus. The long extensors of fingers on both sides yield reduced faradic contractility, and rather sluggish galvanic contraction, through CaCC > AnCC. Legs: Quantitative reduction to faradism and galvanism in calves: CaCC = AnCC. Extensor longus pollicis and extensor longus digit. have almost no faradic contractility, and to galvanism yield slow C, with CaCC = AnCC, the latter almost tetanic. Through external popliteal nerve better contractions (both by faradism and galvanism) are obtained in these muscles. Tibialis anticus and small muscles of feet, normal reactions. In other words, we have in this case a well-marked DeR, limited to the nervo-muscular apparatus of both thumbs; partial DeR in long extensors of fingers and in extensors (and flexors) of toes.

Second Category: Analogous Indigenous Cases.

Case I.—Mrs. X., aged forty years, seen in consultation with Dr. Carreau, of New York, in November, 1885. There is a clear history of excessive drinking and sexual indulgence for many years. Patient is still drinking brandy steadily. For more than two years has suffered from gastric catarrh und failing memory

Early in past summer felt burning sensations in feet, pains in feet and legs; occasional numbness in feet and hands. At end of July marked increase in gastric symptoms, diffused swelling of hands and feet with prickling numbness and a hyperalgesic state of same parts. Was unable to walk. No arthritis. Hands have also been weak. No bladder symptoms. Pains have ceased, but hands and feet are in state of painful formication; dreads to be touched. There is frequent vomiting of food and mucus. Partial dementia, with a few delusions. Urine examined several times; no sign of renal disease. *Examination:* Tongue tremulous and foul. No motor or sensory symptoms in eyes and face. The grasp is feeble, but long extensors and flexors all act. Opposition of thumb is impossible; marked atrophy of radial side of thenar eminences. Examination shows marked reduction in tactile sensibility in tips of all fingers. The hands are œdematous, with dorsum tense. No arthritis. Lower extremities examined seated: feet puffy and tense, but not red; no arthritis. All movements of feet and toes are possible, though feebly done. State of sensibility cannot be well studied, owing to patient's absurd fear of all instruments and manipulations. This also prevents any electrical tests. Patellar reflex abolished (?). Spine not tender. Diagnosis: alcoholic multiple neuritis. Treatment by reduction of brandy, injections of strychnia, galvanism, and massage begun. Carlsbad sprudelsalz for gastric catarrh. A note received from Dr. Carreau, a few days ago, states that faradism did not produce contraction at first, and galvanism was used alone until March, when both currents were employed. In March began to walk, and has steadily improved. Now well, except feebleness of memory, and some numbness.

It is interesting to consider that this and other alcoholic cases bear a certain etiological relation to tropical beri-beri. In both cases we have subjects living upon a relative or actual excess of carbonaceous food, alcohol, or rice, as predisposing, if not efficient causes of neuritis.

The two following cases were published in my essay on "Myelitis of the Anterior Horns" (1874 and 1877) as examples of that affection. Reading them in the light of recent advances in nerve pathology, they now appear to have been cases of multiple neuritis, with dropsical symptoms, making them strongly resemble beriberi or kak-ke. I give them merely in epitome, referring to the original for details.

Case II. (Case XVIII. of essay).—Male, aged forty years, seen in January, 1874. In September severe "cold," with bronchitis, temporary suppression of urine, swelling of face and feet, lasting three to four weeks. Some stiffness and subjective coldness in feet. Relieved by strychnia. October 16th, feet heavy and cold, hands awkward and fingers numb. Increasing loss of power in legs. Physician stated positively that neither this nor the former attack was due to Bright's disease. In November marked anæsthesia and nearly complete paralysis of both legs; thigh muscles remained active; no rectal or vesical symptoms. Median distribution in both hands, anæsthetic; the hands and forearms rapidly wasted. No bedsore or cincture feeling. No cerebral symptoms. Improvement from end of December, when movement appeared in toes.

When examined by me he presented an atrophic paralysis of both hands, with partial anæsthesia (mostly tactile) of finger ends. Toes (except great toes), feet, and legs, can be pretty well moved in bed; legs much atrophied, especially in anterior tibial group of muscles. Feet are partly anæsthetic, but present (as do the legs) marked hyperalgesia. No faradic reaction can be had in both extensor pollicis, and only feeble contraction in thenar eminences. These muscles contract well, however, to galvanism (DeR). Complete recovery occurred by end of summer.

Case III. (Case XIX. of essay).—Male, aged forty-seven years, seen in March, 1884. Large, healthy man. Severe "cold" in October, 1883, with bronchitis, slowly followed by numbness and paresis of limbs. The numbness appeared at about the same time in the ends of the upper and lower extremities. November 10th, walking was possible only with the assistance of a stick or a person, but he could still dress himself. Swelling of the feet, with glossy skin, was marked; by November 14th, subjective coldness of feet. A few days later was unable to sit up in bed; much formication up to the knees, and half-way up the forearms; some anæsthesia (?) of the feet. No head or eye symptoms; bladder and rectum normal. In the middle of January, 1884, intense formication ushered in recovery; gradual improvement in power of legs and hands. Feet remained a little swollen. Some wasting of legs. In January noticed numbness in distribution of left superior maxillary nerve on face. Can now walk with help of cane and crutch.

Examination.—No facial symptoms. Movements of arms and hands good, except that extension of the right fingers is incomplete, and is very feeble on left side, especially for the thumb and index-finger. Palmar muscles emaciated, but not atrophied. Sensibility good. Grasp : right, 26° ; left, 16°. Co-ordinates well. In lower extremities the only paralysis is below the knees. The left foot is but slightly movable ; the right, a little more. Toes are somewhat contracted in flexion. Sensibility to contact is good down to ankle, dull below that, very dull in toes. Toes are numb. No œdema of feet or legs. Urine is dense; but free from any signs of renal disease. Electrical examination shows very great loss of faradic contractility in palsied muscles, almost complete in left extensor indicis, and in extensors of toes and flexors of feet (anterior tibial groups) ; feeble reaction in calves. All these muscles, however, react well to galvanism (DeR.).

These three indigenous cases of multiple neuritis—one clearly of alcoholic origin, the others of unknown etiology—I submit with but little comment. They were characterized by a combination of paræsthesiæ and anæsthesia, by paralysis with atrophy and DeR., of irregular peripheral distribution, and by œdema of the extremities, without signs of renal disease. Bronchitis occurred a short time before the nervous symptoms appeared in the two non-alcoholic cases. Cardiac and respiratory symptoms, indicating neuritis of the vagi and the phrenic nerves, were the only ones wanting to produce a symptom-group almost exactly like that of severe beri-beri or kak-ke.

DR. H. C. WOOD made some remarks on

THE DIAGNOSIS OF NEURITIS

and showed two illustrative cases.

DR. CHARLES K. MILLS then read some remarks on

THE CONCURRENCE OF MULTIPLE NEURITIS WITH MYELITIS OR ENCEPHALITIS.

As one result of the interest which has been aroused in the subject of multiple neuritis, or polyneuritis, some cures have been effected which were before not considered possible ; but it seemed to the speaker that, as in the case of other advances in medicine, some have been led to go too far in their conclusions, and have overlooked facts which are worthy of close attention. That multiple neuritis frequently occurs, can no longer be doubted ; but that it occurs frequently as an absolutely isolated affection is, to say the least, doubtful. His own experience was, that not infrequently either myelitis or encephalitis, or both, concur with this affection; that is, in not a few cases in which the diagnosis of multiple neuritis could be made by the strict rules applicable to the study of this affection—by a certain combination of motor and sensory symptoms, by their localization in distal parts of the limbs, and by tenderness of the nerve trunks, and of the muscles—positive evidences of the existence of the central disorders were also present.

If, as is generally believed, multiple neuritis is the result of a direct toxic influence on the nerve elements themselves, it was reasonable to suppose that this same influence would frequently be exercised upon the nerve elements of the brain and spinal cord.

Dr. Mills' conclusions were that we may have four classes of cases: 1. Cases of multiple neuritis, pure and simple; such cases, however, were rare. 2. Cases in which the multiple neuritis was associated with myelitis. 3. Cases in which multiple neuritis was associated with encephalitis. 4. Cases in which multiple neuritis was associated with both myelitis and encephalitis. Instead of the association being just as is indicated by this classification, the combination might be of neuritis or peri-neuritis, with meningo-myelitis or meningo-encephalitis; or, finally, any modification of these combinations might be found.

Dr. Mills referred to a case which he had seen in consultatior recently, in which the patient presented all the typical marks o multiple neuritis, the condition of her limbs being such as to call to mind the picture in Gowers' recent book of a case of multiple alcoholic neuritis, with palsy of extensors of wrist and flexors of ankle. This patient had also very positive evidences of cerebral involvement—mental confusion, with hallucinations and delusions both of sight and hearing. She had a clear history of abuse, not only of alcohol, but also of opium, chloral, and other narcotics. He believed that in this case there was a concurrence of multiple neuritis with a similar pathological condition in the spinal cord and brain. He also referred to other cases, seen in consultation, and more particularly in the Philadelphia Hospital, where the majority of patients have been the victims of alcoholic excess—cases in which a confused picture of nerve, cord, and cerebral disease, probably inflammatory, was frequently presented. Cases of poisoning by lead, mercury, and particularly arsenic, were also discussed. Particular reference was made to the series of cases of poisoning by arsenic which he reported to the College of Physicians of Philadelphia in 1883.

A most interesting case, as bearing upon the views here presented, was one which he saw with Dr. O'Hara, of Philadelphia, to whom he was indebted for notes of the past history of the case, and also for suggestions as to the condition present at different stages. This patient, a professional man of originally good physique, and of remarkable intellectual attainments, had, eight years ago, an attack of rheumatism, which left him with a crippled heart, the damage to this organ, however, not seeming to have been of its valvular, but rather of its nervous structures. The patient was in the habit of using tobacco and alcohol, but not to excess, and sometimes did not use these agents at all for long periods. Two years after the first attack of rheumatism he began to have trouble with his vision. Sight in both eyes slowly failed until he scarcely had more than perception of light in both eyes. Careful ophthalmoscopic examinations were made with negative results; the media were clear, and no signs of retinitis or neuritis were discovered. In eighteen months he entirely recovered his sight from travel, rest, and the use, in the latter months, of strychnia hypodermatically injected. About a year after recovery of his sight he had another attack, rheumatic or rheumatoid gouty in character. In this attack he had insomnia and evidences of general nervous irritability. At this time the left upper extremity suffered from an undoubted attack of neuritis, affecting the most of the brachial nerves, but more markedly those of the median distribution.

About six months ago, he had what appeared to be an appoplectiform attack, his left side—face, arm, and leg—being temporarily paralyzed, but without unconsciousness. The attack passed off in a few hours.

About two months since he was engaged in work which called for both mental and physical effort, and considerable exposure. One day, when at a height of more than one hundred feet, he was suddenly stricken with incomplete paralysis of motion and sensation in the lower extremities, accompanied by great pain, but succeeded by his own efforts in getting again to the ground. This attack he attributed, probably correctly, to a stroke of air. On examination, he was found to have almost complete paralysis of the left leg from the knee downward, with blueness and coldness of the foot, and typical anæsthesia dolorosa as high as the ankle. He complained of excruciating pains, which he described as "jamming" in the sole of the foot. The right leg showed a similar condition, but much less marked, the paralysis of motion and sensation extending only half-way to the knee, and not being nearly as complete as upon the other side. In two weeks, under the use of anodynes, local and general, with massage and weak currents of electricity, he recovered completely from these symptoms in his limbs. He then began again to use both head and limbs, without rhyme or reason, and was attacked with loss of power and great pain, and perverted sensation in the right leg below the knee, with swelling of the dorsum of the foot and of the ankles. He also began to have lightning pains, here and there, in both lower extremities, and radiating from the spine upward, these being sometimes accompanied by retraction of the head. Still later he developed symptoms of cerebral excitement—total insomnia, refusal to take medicine, quasi-delusions, great loquacity, etc. He was without sleep for nine days and nights. Various remedies were vainly tried, but the first genuine relief which he obtained was from hypodermic injections of morphia, repeated every twelve hours. This was followed by the administration of deodorized tincture of opium regularly every four hours, in doses of from ten to twenty mimims, with twenty minims at bedtime. This treatment was continued, with digitalis, which he had been taking more or less for six or seven years, for about nine days. Mild alcoholic stimulus was also used, and nourishment was systematically administered. He has improved to such an extent that all the symptoms, neural, spinal, and cerebral, have greatly diminished, so that he is able to travel, but he cannot yet endure any great strain, either physical or mental.

DR. JAMES HENDRIE LLOYD read :

NOTES ON THE ELECTRO-DIAGNOSIS OF NEURITIS AND ANTERIOR POLIOMYELITIS.

The irritability of a nerve-trunk to an electric current (either by galvanism or faradism) depends upon the integrity of the axis-cylinder. This integrity may be impaired by pressure upon or by a fracture of the axis-cylinder. The pathology of degeneration in a nerve-fibre is, first, an increase of protoplasm about the nucleus in the sheath, then the splitting up of the white matter, and, finally, the breaking of the axis-cylinder. In neuritis it may be a question how soon this *fracture* takes place, but *pressure* evidently occurs very early, and in severe acute neuritis (as in some forms of facial paralysis) this pressure is very great, is very quickly established, and, in the worse forms, no doubt leads to the breaking of the axis-cylinder. If electro-irritability, therefore, be retained in the nerve in neuritis, it must be either because the pressure is very slight or else because of an escape of some nerve-fibres amidst the destruction of others. The first event is what happens in mild subacute neuritis, but the latter alternative does not seem very probable when the disease is interstitial in the narrow calibre of a nerve-trunk, and especially when it has continued a long while. On the other hand, in anterior poliomyelitis of slow progress the large cells may be gradually and successively destroyed, followed by the degeneration of a comparatively few nerve-fibres—and in these cases the nerve will retain its electro-irritability (possibly diminished sometimes) although the muscles may have commenced to put on the modal and serial changes of degeneration. These facts have been long recognized, but do not appear of late to have been sufficiently relied upon as a possible factor in diagnosis.

De Watteville has drawn a distinction between myotrophic and neurotrophic centres in the cord, but the distinction, as well as his illustration, is artificial, and the phenomena probably depend upon the extent of degeneration and the number of nerve fibres involved. His own opinion inclines to the view that where nerve-irritability (especially faradic) is preserved, especially in cases otherwise chronic, with muscular atrophy and muscular reactions of degeneration, we have a condition rather of atrophy of the anterior horns than of the nerve-trunks, affording strong corroborative evidence in establishing the differential diagnosis. It is this condition of retained nerve-irritability, with degeneration reactions to galvanism in the muscle, which we have in such slow cord-lesions as amyotrophic lateral sclerosis and chronic anterior poliomyelitis. Here other symptoms aid the diagnosis, especially the large fibrillary contractions in the muscular atrophy and the spastic symptoms in lateral sclerosis. The only forms of neuritis that he knew of—in which faradic irritability of the nerve is preserved, with muscular reactions of degeneration—are essentially mild, non-chronic attacks (such as mild facial palsy), and in these the lesion is probably light compression, leaving the axis-cylinder unbroken.

It must be recalled that muscle-tissue alone will not respond to such strength of faradism as we use upon it in the clinic, but is excited through the motor nerve-endings in its tissue, consequently when we have response in a muscle to faradism we infer that the nerve is still intact or partially so. Hence in severe facial palsy one of the first symptoms is loss of farado-contractility.

Illustrative cases were cited.

Conclusions.—The point sought to be emphasized is the retention of electro-excitability in the nerves, which in chronic cases does not appear consistent with the idea of a neuritis, but, rather, of a slow cord-lesion. If this electric excitability can be preserved in a chronic neuritis, we must suppose a very slow interstitial inflammatory process which compresses some fibres and allows others to escape. But to think that this happens in the narrow calibre of a nerve-trunk during a prolonged period of inflammation seems to require some effort of imagination and credulity. The electrical reactions in the above cases (especially the condition of *nerve-*irritability) support the other symptom-groups in their significance as to a cord- or a nerve-lesion.

Dr. H. C. Wood stated his belief that chronic multiple neuritis is one of the most frequent of the severe organic nerve diseases, and that in a large proportion of cases of serious nerve disease, the whole spinal axis and its accessories are affected. In general paralysis of the insane, four-fifths of the cases have some lateral or posterior spinal sclerosis, and a large proportion of these cases, no doubt, have multiple neuritis. He thinks that it is perfectly proven that locomotor ataxia may travel down the cord and produce peripheral neuritis, and that it is equally well proven that peripheral neuritis may travel upward, producing secondary lesions in the cord. With reference to electrical studies, it may be said that a large number of cases show that in the various forms of neuritis every possible change in the electrical reactions can be found. Post-mortem examinations are sufficiently numerous to show that peripheral neuritis is frequently not associated with other diseases, and that it frequently causes symptoms which simulate other affections. When we have peripheral neuritis, it is difficult to make out a spinal trouble back of it.

Dr. Osler thought that it was clear that we have the curious disease beri-beri in this country. It is probable that it exists in the Gulf States, as, a few months ago, he received some slides, which had been sent by a physician of Texas, and were said to contain the micro-organism of beri-beri. Any one reading the descriptions of this disease must have been struck with the remarkable resemblance of some of the cases to acute ascending paralysis. His friend, Dr. Vineberg, now of New York, who formerly practised in the Sandwich Islands, gives a very accurate account of the suddenness of onset, the rapidity of the ascension of the paralysis, which, in some cases, ultimately involves the respiratory muscles, producing death in that way. He agreed with Dr. Wood that multiple neuritis must be an exceedingly common disease. The French journals of the current year contain a surprising amount of literature on the subject. He had been particularly interested in cases of neuritis occurring in connection with certain of the fevers. After typhoid fever it has been described a number of times, and also after phthisis. During the past year he has had one interesting case of paralysis of the extensors of the left foot in connection with phthisis, unquestionably a case of neuritis. He suggested that possibly some of these cases of paræsthesia of the hands, with feelings of numbness and tingling, which Putnam has described so accurately, may be of the nature of a peripheral neuritis. A case recently presented itself at his service at the Infirmary for Nervous Diseases, in which these symptoms were very marked, and in which there apparently were some trophic changes. The skin was glossy, and the hands were somewhat swollen, and, indeed they presented in a slight degree the appearances seen in myxœdema.

In connection with the diagnosis between acute ascending paralysis, anterior myelitis, and multiple neuritis, he would refer to an article by Francotte in the *Revue de Médecine*, to which attention was called in *The Medical News* of July 3d, 1886. He reports four cases, two of which were studied anatomically, and in which the cord was found to be absolutely normal. In these two cases, the clinical picture was that of Landry's ascending paralysis.

DR. SEGUIN said that he was much interested in the remarks of Dr. Mills as to the possible co-existence of a central with a peripheral lesion. He had no doubt that this co-existence is frequently a source of considerable difficulty in making up our minds whether a case is one of irregular poliomyelitis or of peripheral neuritis. It also seemed to him to be a matter of great practical importance as regards treatment that we should make up our mind whether we have a case of perineuritis or one of parenchymatous neuritis. The former disease, which is, he thinks, the more common, and perhaps present in some cases of so-called neuralgia, is essentially an inflammatory change in the connective tissue, perhaps penetrating into the interior of the nerve bundles, but not affecting the nutrition of the axis-cylinder or myelin and not capable of producing any distinct paralysis or atrophy. The diagnosis may be made clear by relying on this distinctive point, that in interstitial neuritis there must be anæsthesia, even if limited in area ; and a true paralysis and atrophy, even if restricted to a few muscles. In perineuritis the symptoms are more diffused, milder, more subjective, and do not present a specific appearance. He thought that Dr. Lloyd had struck the key-note in insisting on the importance of a careful study of the neural and muscular reactions. In true neuritis and interstitial neuritis, there must be degenerative reactions in some shape, distal from the point of disease, while in perineuritis and in some forms of myelitis the reactions are either irregular or simply a quantitative or nodal reduction.

The diagnosis is of great importance with respect to treatment. Perineuritis and meningitic conditions he treats with great energy with counter-irritation, with the actual cautery and blisters. Internally, he employs mostly tonic treatment, and does not resort to mercury or iodide of potassium unless there is a special reason for their use. Rest is also an important element in the treatment of perineuritic conditions. In essential neuritis, therapeutic measures can have but little effect, as we know that a natural repair takes place irrespective of artificial conditions. If we favor nutrition by our general remedies, that is the most that we can do, although galvanism may serve to keep up a better nutrition in muscles pending nerve regeneration.

In regard to the relation between neuritis and beri-beri, he did not wish to be understood as stating positively that we have beri-beri among us, but he thought that it would not be out of place to call attention to the analogy between the symptoms of the two affections. He thought it well to have the attention of physicians in our Gulf States called to the features of the disease. As the disease has recently appeared in northern Brazil, it may appear in our Southern States.

(To be continued.)